

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Attorney Docket No.: **ISPH-0756**

Inventors: **Bennett and Dobie**

Serial No.: **Not Yet Assigned**

Filing Date: **Herewith**

Examiner: **Not Yet Assigned**

Group Art Unit: **Not Yet Assigned**

Title: **Antisense Modulation of Superoxide
Dismutase 1, Soluble Expression**

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By *Jane Massey Licata*
Typed Name: **Jane Massey Licata, Reg. No. 32,257**

Commissioner for Patents
Mail Stop Sequence
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

INFORMATION DISCLOSURE STATEMENT

Pursuant to 37 C.F.R. §1.56 and in accordance with 37 C.F.R. §§1.97-1.98, information relating to the above-identified application is hereby disclosed. Inclusion of information in this statement is not to be construed as an admission that this information is material as that term is defined in 37 C.F.R. §1.56(b).

(XX) In accordance with §1.97(b), since this Information Disclosure Statement is being filed either within three months of the filing date of the above-identified application, within three months of the date of entry into the national stage of the above identified application as set forth in §1.491, or before the mailing date of a first Office Action on the merits of the above-identified application, no additional fee is required.

() In accordance with §1.97(c), this Information Disclosure Statement is being filed after the period set forth in §1.97(b) above but before the mailing date of either a Final Action under §1.113 or a Notice of Allowance under §1.311, therefore:

() Certification in Accordance with §1.97(e) is set forth below; or

() The fee of \$240.00 as set forth in §1.17(p) is attached.

() In accordance with §1.97(d), this Information Disclosure Statement is being filed after the mailing date of either a Final Action under §1.113 or a Notice of Allowance under §1.311 but before the payment of the Issue Fee, therefore included are: Certification in Accordance with §1.97(e); Petition Requesting Consideration of the Information Disclosure Statement; and the fee of \$130.00 as set forth in §1.17(I)(1).

() Copies of each of the references listed on the attached Form PTO-1449 (modified) are enclosed herewith.

(XX) In accordance with §1.98(d), copies of some or all of the references listed on the attached Form PTO-1449 (modified) are not enclosed herewith because they were previously submitted to the U.S. Patent and Trademark Office in prior application Serial No. 09/888,360, filed June 21, 2001 for which a claim for priority under 35 U.S.C. §120 has been made in the instant application.

Please charge any deficiency or credit any overpayment to Deposit Account No. 50-1619. This form is submitted in duplicate.

() The relevance of the listed references in a foreign language is as stated in the specification at pages @@.

(XX) All listed references are in the English language.

Respectfully submitted,

Jane Massey Licata

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Date: August 4, 2003

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Form PTO-1449 Modified		Docket No. ISPH-0756	Serial No. not yet assigned
List of Patents and Publications Cited by Application (Use several sheets if necessary) -- U.S. Department of Commerce Patent and Trademark Office		Applicant C. Frank Bennett et al.	
		Filing Date herewith	Group not yet assigned
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)			
	AA	Al-Chalabi et al., Recent advances in amyotrophic lateral sclerosis, Curr. Opin. Neurol., 2000, 13:397-405	
	AB	Alisky et al., Gene therapy for amyotrophic lateral sclerosis and other motor neuron diseases, Hum. Gene Ther., 2000, 11:2315-2329	
	AC	Bruijn et al., Aggregation and motor neuron toxicity of an ALS-linked SOD1 mutant independent from wild-type SOD1, Science, 1998, 281:1851-1854	
	AD	Cleveland et al., Oxidation versus aggregation - how do SOD1 mutants cause ALS?, Nat. Med., 2000, 6:1320-1321	
	AE	Fridovich, Superoxide radical and superoxide dismutases, Annu. Rev. Biochem., 1995, 64:97-112	
	AF	Gulesserian et al., Superoxide dismutase SOD1, encoded on chromosome 21, but not SOD2 is overexpressed in brains of patients with Down syndrome, J. Investig. Med., 2001, 49:41-46	
	AG	Hottinger et al., The copper chelator d-penicillamine delays onset of disease and extends survival in a transgenic mouse model of familial amyotrophic lateral sclerosis, Eur. J. Neurosci., 1997, 9:1548-1551	
	AH	Kawata et al., Aberrant splicing of human Cu/Zn superoxide dismutase (SOD1) RNA transcripts, NeuroReport, 2000, 11:2649-2653	
	AI	Klivenyi et al., Neuroprotective effects of creatine in a transgenic animal model of amyotrophic lateral sclerosis, Nat. Med., 1999, 5:347-350	
	AJ	Lee et al., Molecular cloning and high-level expression of human cytoplasmic superoxide dismutase gene in Escherichia coli, Misaengmul Hakhoechi, 1990, 28:91-97	
EXAMINER		DATE CONSIDERED	

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		Filing Date herewith _	Group not yet assigned
U.S. Department of Commerce Patent and Trademark Office			
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)			
	AK	Rothstein et al., Chronic inhibition of superoxide dismutase produces apoptotic death of spinal neurons, Proc. Natl. Acad. Sci. U. S. A., 1994, 91:4155-4159	
	AL	Rowland, Six important themes in amyotrophic lateral sclerosis (ALS) research, 1999, J. Neurol. Sci., 2000, 180:2-6	
	AM	Trotti et al., SOD1 mutants linked to amyotrophic lateral sclerosis selectively inactivate a glial glutamate transporter, Nat. Neurosci., 1999, 2:427-433	
	AN	Troy et al., Downregulation of Cu/Zn superoxide dismutase leads to cell death via the nitric oxide-peroxynitrite pathway, J. Neurosci., 1996, 16:253-261	
	AO	Troy et al., Down-regulation of copper/zinc superoxide dismutase causes apoptotic death in PC12 neuronal cells, Proc. Natl. Acad. Sci. U. S. A., 1994, 91:6384-6387	
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U.S. PATENT DOCUMENTS

Examiner's Initial		Document No.	Date	Name	Class	Subclass
	AA					
	AB					
	AC					
	AD					
	AE					
	AF					
	AG					
	AH					
	AI					
	AJ					
	AK					
	AL					
	AM					
	AN					

FOREIGN PATENT DOCUMENTS

Examiner's Initial		Document No.	Date	Country	Translation YES NO	
	AO	Wo 94/19493	9/1/1994	PCT	X	
	AP					
	AQ					
	AR					
	AS					
	AT					
	AU					
	AV					
	AW					
	AX					

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